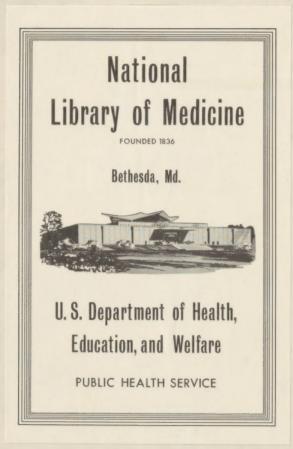
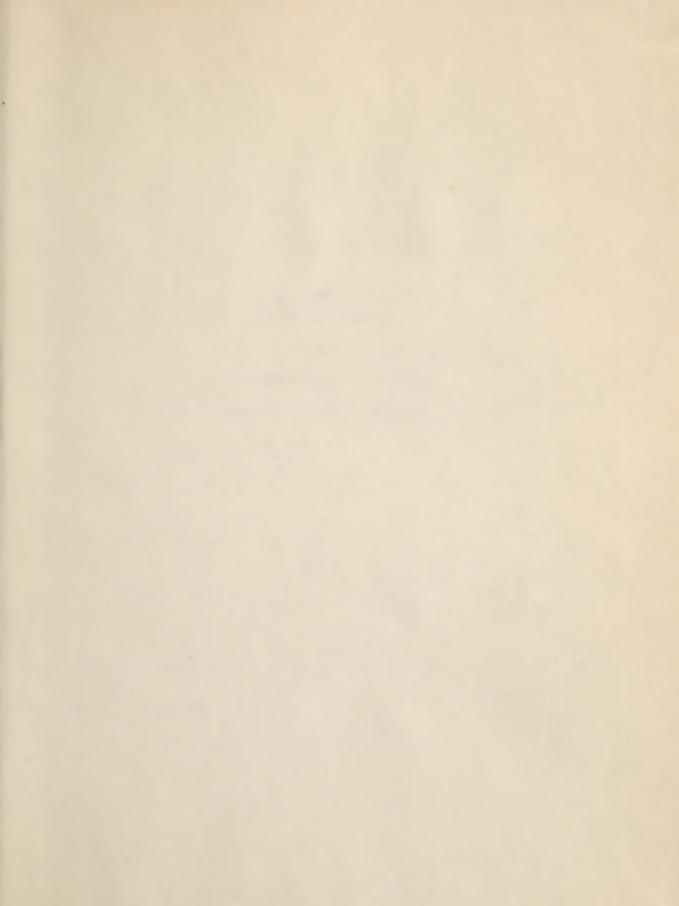
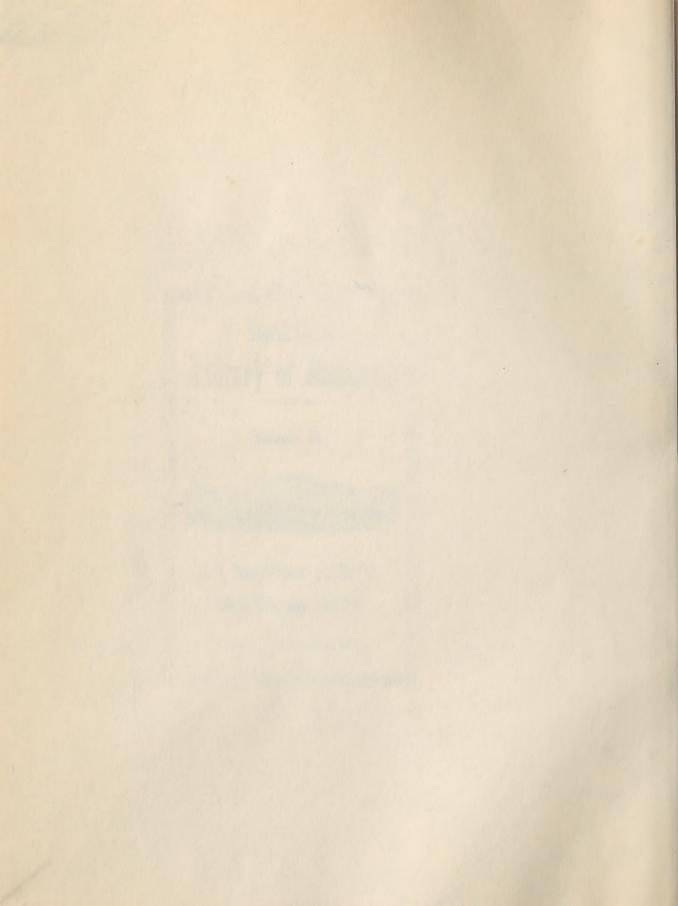
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GASTROENTEROLOGY SECTION

U.S. Army. BROOKE GENERAL HOSPITAL, Fort Sam Houston, Texa.

1948 1949

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Legend for Pictures

Brooke General Hospital. Main Building. Top

Parasitology technician examining a direct Upper Left smear scraping from procto-sigmoidoscopic

examination.

Procto-sigmoidoscopic examination. Upper Right

Lower Left Gastroscopic examination.

Lower Right Diagnostic biliary drainage- Lyon technic.











Legend for Pictures

Top Brooke General Hospital -- Annex IV

Upper Left Patients' lounge-- Ward 25

Opper Right Nurse's office - Ward 25

Lower Left Gastro-intestinal hemorrhage cubicle- Ward 25

Lower Right Ward 25











SECTION FACILITIES

During the period of this report, the Gastroenterology Section has consisted of one 55 bed male ward in Annex IV of the Brooke General Hospital, a clinic room in the same Annex, and the offices of the Chief of Section for consultation visits of patients and administration of the Section.

The ward is a series of adjacent cubicles and contains no private rooms. Each bed can be curtained off when desired. One 40 bed part of the ward is separated by a roomy patients' lounge room, and the ward and section offices from the 15 bed balance of the ward. Extending from the larger ward space to the smaller is the Nurse's office, Section office, utility room, linen closet, and Ward Officer's office. An adequate and modern latrine is a part of the ward. During 1948, a part of this was isolated for the use of patients with infections or infestations, such as dysentery or amebiasis. Patients in this category are constantly educated to use the isolation facilities.

The Gastroenterology Section Clinic in room 230 Annex IV was formerly used as an operating room. In this room were installed five hospital beds, a gastroscopic table, as Lane procto-sigmoidoscopy table, suction apparatus, miscellaneous equipment for clinical microscopy, and a sub-laboratory. In this Clinic room are performed the gastroscopic examinations, procto-sigmoidoscopies, diagnostic biliary drainages, and fractional gastric analyses.

On 8 December 1947, when the present Chief of Section first assumed his duties, there were no standing operative procedures on the Section. During 1948 the following procedures were originated and have been in use during 1949.

- 1. Management of massive gastro-intestinal hemorrhage.
- 2. Routine feces examination.
- 3. Liaison with Fourth Army Area Medical Laboratory in cases of amebiasis.
- 4. Preparation of patient for infestation study of feces.
- 5. Preparation of patient for fractional gastric analysis.
- 6. Preparation of patient for diagnostic biliary drainage.
- 7. Preparation of patient for procto-sigmoidoscopy.
- 8. Preparation of patient for gastroscopy.
- 9. A gastro-intestinal history check sheet for Residents.

10. Many gastro-intestinal special diets.

11. Combined intensive therapy for portal cirrhosis.

12. Lyon technic for diagnostic biliary drainage.

13. Modified Rehfuss technic for fractional gastric analysis.

14. Technic for Winklestein drip treatment.

II

EDUCATION

1. Residents in Internal Medicine

Each Resident in internal medicine, during his three year training period, serves two months on the gastro-enterology section. During this training period, the emphasis is placed on basic clinical gastro-enterology, such as the proper method of taking a good gastro-intestinal history, the technic of physical disgnosis as applied to the gastro-intestinal tract, a study of the technics of the common gastro-intestinal diagnostic procedures, such as fractional gastric analysis, proctosigmoidoscopy, diagnostic biliary drainage, and gastroscopy. He is also trained in the special therapeutic procedures carried out on ward 25.

The Resident on the gastroenterology section is also the Ward Officer for Ward 25. All of the administrative duties peculiar to those of a ward in an Army General Hospital are his direct responsibility. Under his authority are personnel including an intern, three to five nurses, five to six ward attendants covering the 24 hour period.

In addition to his duties on the Gastroenterology Section, he must attend other forms of resident training in the course of Internal Medicine. Some of these are the clinico-pathological conference, the disgnostic medical conference, medical literature review staff meetings, the x-ray conference, the tumor board, and all special clinics given for the Medical Service by visiting consultants through-out the year. He attends all autopsies performed on patients dying on Ward 25, and others as his time permits.

Each resident receives a copy of "An Outline of Gastroenterology", written by the Chief of Section, as a reference manual during his training and for his future use. The journal 'Gastroenterology' is abstracted currently and made in manual form for reference. The Section library has adequate text material.

Bedside teaching and informal discussions have been the rule. Each Wednesday forenoon, several interesting cases on Ward 25 are presented to the consultant physician by the personnel of the gastroenterology section. These are of the teaching grand round type.

During 1948 and 1949 the following lectures, clinics, and academic staff meetings have occurred.

- 1) Clinics by Dr. Verne R. Mason (Special Consultant to The Surgeon General in Internal Medicine at Brooke General Hospital).
 - a. 5 January 1948

 Two cases of duodenal diverticulum with peptic ulcer in the diverticulum.
 - b. 7 January 1948
 Case of gastric ulcer
 Case of hematemesis with epileptic seizures
 Case of anorexia nervosa
 Case of abdominal tumor
 Case of esophageal varix
 Case of sub-hepatic abscess-cause undetermined
- 2) Medical Diagnostic Conference 23 January 1948
 Case of amebic hepatitis
 Presented by Lt. A. F. Mann
- 3) Medical Diagnostic Conference 31 January 1948
 Case of syphilis of the stomach
 Presented by Capt. H. Grant
- 4) Medical Diagnostic Conference 13 March 1948
 Case of psychogenic gastro-intestinal
 reaction, manifested by chronic diarrhea.
 Presented by Capt. T. W. Inmon
- 5) Medical Diagnostic Conference 23 March 1948 Case of obstruction of the common bile duct with possible obstruction of the pancreatic duct.

 Presented by Capt. H. Grant
- 6) Medical Diagnostic Conference 3 April 1948
 Case of hemolytic jaundice
 Presented by Capt. F.M. Groat
- 7) Medical Diagnostic Conference 24 April 1948
 Case of carcinoma of the right colon with possible cirrhosis of the liver.
 Presented by Lt. F. B. Graves

- 8) Medical Literature Review 17 May 1948
 Biochemistry of fat soluble vitamins.
 Presented by Capt. B.I. Copple
- 9) Medical Staff Conference 18 May 1948
 Biochemistry of fat soluble vitamins-Part II
 by Capt. B.I. Copple
- 10) Medical Staff Conference 25 May 1948
 Water soluble vitamins-Part I
 by Capt. W.C.Winter and Capt. G.F.Horton
- 11) Medical Literature Review 14 June 1948
 Water soluble vitamins- Part II
 by Lt. R. D. Wright and Capt. G. E. Horton
- 12) Medical Staff Conference 15 July 1948
 Leptospirosis
 by Capt. T. W. Inmon
- 13) Medical Literature Review 19 July 1948
 Biochemistry of Digestion-Part I
 by Lt. Col. Augustus A. Hall
- 14) Medical Staff Conference 22 July 1948
 Functional diseases of the esophagus
 by Lt.Col. Augustus A. Hall
- 15) Medical Staff Conference 29 July 1948
 Functional diseases of the stomach
 by Lt. Col. Augustus A. Hall
- 16) Medical Literature Review 2 August 1948
 Biochemistry of Digestion-Part II
 by Lt. Col. Augustus A. Hall
- 17) Medical Staff Conference 5 August 1948
 Peptic Ulcer
 by Lt. Col. Augustus A. Hall
- 18) Medical Staff Conference 12 August 1948
 Organic Diseases of the Stomach
 by Lt. Col. Augustus A. Hall
- 19) Medical Staff Conference 30 September 1948
 Acute pancreatic necrosis
 by Lt. Col. Augustus A. Hall and Lt.Col.T.D.Johnson
- 20) Medical Diagnostic Conference 2 October 1948
 Case: Differential diagnosis between adenocarcinoma
 of fundus of stomach and Hodgkin's disease.
 by Major K. D. Orr

- 21) Monthly Staff Conference-Brooke General Hospital
 19 October 1948
 Combined Intensive Treatment of Cirrhosis of Liver
 by Lt. Col. Augustus A. Hall
- 22) Medical Literature Review 6 December 1948
 Regional Enteritis
 by Major L.F. Parmley
- 23) Medical Staff Conference 16 December 1948
 Psychosomatic Medicine
 by Major H. E. Ratcliffe
- 24) Medical Diagnostic Conference 19 December 1948
 Case of chronic ulcerative colitis
 Presented by Major L.F. Parmley
- 25) Medical Diagnostic Conference 5 February 1949
 Case: Differential diagnosis of gastric carcinoma
 and peptic ulcer, hemorrhage-site undetermined.
 Presented by Major T. W. Inmon
- 26) Medical Staff Conference 10 February 1949
 Hepatitis
 by Major T.W.Inmon
- 27) Medical Diagnostic Conference 12 February 1949
 Case: Differential diagnosis between chronic
 cholecystitis and infectious mononucleosis and
 radiculitis.
 Presented by Major T. S. Malinowski
- 28) Clinic on gastroenterology by Dr. Garfield G. Duncan
 14 February 1949
 Two cases of infectious hepatitis
 Case of homologous serum hepatitis
 Case of cirrhosis with hepatic failure
 Case of cirrhosis with good response to therapy
 Case of Laennec's cirrhosis with good response
 to therapy, checked by biopsy.
- 29) Medical Staff Conference 17 February 1949
 Lecture on hepatitis
 by Dr. Garfield G. Duncan
- 30) Medical Diagnostic Conference 19 February 1949 Case: Gastric ulcer Presented by Capt. R. D. Wright

- 31) Medical Staff Conference 3 March 1949 Cholecystitis and cholelithiasis by Capt. R. W. Blohm
- 32) Medical Staff Conference 10 March 1949 Constipation by Major Charles J. Hornisher
- 33) Medical Diagnostic Conference 26 March 1949 Case: Anorexia nervosa. Presented by Capt. R. D. Wright
- 34) Monthly Staff Conference-Brooke General Hospital 3 May 1949 Lecture on Cancer of the Stomach by Dr. B. R. Kirkland, Mayo Clinic
- 35) Medical Literature Review

 Moving Picture Films

 'Role of gastroscopy in the diagnosis and treatment of gastric pathology'

 'Vagotomy for duodenal ulcer'

 'Human small intestine— study on motility'

 Presented by Lt. Col. Augustus A. Hall
- 36) Medical Diagnostic Conference 28 May 1949
 Case: Differential diagnosis between aplastic
 anemia, Laennec's cirrhosis of the liver following
 chronic hepatitis and hypersplenism.
 Presented by Major H. E. Ratcliffe
- 37) Medical Diagnostic Conference 4 June 1949
 Case: Filariasis.
 Presented by Major K. D. Orr
- 38) Medical Diagnostic Conference 6 August 1949
 Case: Weil's disease
 Presented by Capt. R. D. Gamble
- 39) Medical Diagnostic Conference 20 August 1949
 Case: Liver abscess
 Presented by Major K. D. Orr
- 40) Clinics on gastroenterology by Dr. James A. Greene and Dr. Don W. Chapman.**

a. 29 August 1949 Morning-

Case of Laennec's cirrhosis of the liver.

Case of chronic pancreatitis
Case of duodenal ulcer
Case of chronic hepatitis, post-splenectomy.
Case of gastric ulcer

Afternoon-

Case of hemochromatosis
Case of amebic abscess of the liver.

- 41) Medical Diagnostic Conference 10 September 1949
 Case: Chronic relapsing pancreatitis
 Presented by Major K. D. Orr
- 42) Medical Literature Review 3 October 1943
 Current therapy in gastrointestinal diseases
 by Capt. R. D. Gamble
- 43) Medical Staff Conference 8 October 1949
 Pancreatic diseases
 by Capt. R. D. Wright
- 44) Medical Diagnostic Conference 10 December 1943
 Case: Hysterical non-gaseous distention of the abdomen.
 Presented by Capt. J. B. Crow
- 45) Medical Diagnostic Conference 6 October 1949
 Case: Regional enteritis
 Presented by Lt. Col. W. S. Bagnall
- 46) Medical Staff Conference 15 December 1949
 Intestinal parasites
 by Lt. Col. J. L. Tobin

The academic meetings listed above were on gastroenterologic subjects. They represent only a fraction of the total number of similar meetings on the entire field of internal medicine during the two year period of this report. All of these were conducted by the Medical Service under Colonel John G. Knauer, Chief of the Medical Service.

III

PERSONNEL

The following personnel have served on the Gastroenterology Section during 1948 and 1949.

1. Teaching Staff

** Col. John G. Knauer, M.C. Chief of the Medical Service

* Lt. Col. Augustus A. Hall M.C. Chief, Gastroenterology Section

** Dr. David R. Sacks Civilian Consultant

** Dr. James A. Greene Civilian Consultant

** Dr. Don W. Chapman Civilian Consultant

** Dr. Don W. Chapman Civilian Consultant

** Dr. Henry N. Leopold Civilian Consultant

** Dr. William W. Bondurant Civilian Consultant

2. Residents in Internal Medicine

Lt. Col. T. D. Johnson | 1 January 29 February 1948

Capt. T. W. Inmon 1 March- 30 April 1948

Major A. F. Nancekivell 1 May- 15 June 1943

Major C. J. Hornisher 1 May - 30 June 1948
Major W. H. Crosby 1 July - 31 August 1948

Capt. R. W. Blohm 1 September 31 October 1948

Major L. F. Parmley 1 November - 31 December 1948

Major W.H. R. Croskery 1 January 28 February 1949 Lt. Col. T. E. Huber 1 March 30 April 1949

Lt. Col. T. E. Huber 1 March- 30 April 1949
Major H. E. Ratcliffe 1 May- 31 August 1949

Capt. J. F. Stagg 1 July - 31 July 1949

Major K. D. Orr 1 August 31 October 1949

Lt. Col. J. L. Tobin 1 November- 31 December 1949

3. Interns

1st Lt. William C. Hartley 1 July - 31 July 1949

1st Lt. Ray E. Yarbrough 1 August - 31 August 1949

1st Lt. Donald D. Jacobson 1 September 30 September 1949

1st Lt. Alford R. Hazard 1 October 31 October 1949

1st Lt. James A. Nesse 1 November - 30 November 1949

1st Lt. Robert F. Morgan 1 December 31 December 1949

4. Army Nurse Corps

Name Rank

Anna K. Schelper Capt.
Frankie D. Harris Capt.
Mary C. Axmann Capt.
Hazel Cooley Capt.
Eva E. Cheeseman Capt.
Suzanne N. Ottoy lst Lt.
Evalyn Swanson lst Lt.

Evalyn Swanson lst Lt.

Amymae H. Young lst Lt.

* Fellow, American College of Physicians

** Diplomate, American Board of Internal Medicine

Kathleen Taylor	lst	Lt.
Doreen J. Harrell	lst	Lt.
Elizabeth L. Dietz	2nd	Lt.
Betty Jo Phillips	2nd	Lt.
Mary V. Lane	2nd	Lt.
Josephine Kovach	2nd	Lt.
Dorothy L. Meetze	2nd	Lt.
Corinne N. Salvucci	2nd	Lt.
Janet F. Holman	2nd	Lt.

5. Civilian Nurses

Name	Rank
Laura K. Hughes	R.N.
Louise 5. Ashburn	R.N.
Elizabeth A. Dunnaway	R.N.
Zora L. Ezzell	R.N.
Imogine Frazer	R.N.
Elizabeth A. Howell	R.N.
Velma L. Kerschner	R.N.
Helen L. St. Martin	R.N.
Lillian E. Silvers	R.N.
Dorothy D. Thompson	R.N.
Mabel S. Wolfe	R.N.

6. Enlisted Personnel

Name

Merino	Theyeare
Walter L. Davis	Sgt., lst Cl.
John R. Phillips	Sgt., 1st Cl.
John O. Bryant	Sgt.
Elmo C. Wade	Sgt.
Jesus M. Martinez	Sgt.
George M. Barron	Sgt.
F. Phillips	Sgt.
Ignacio V. Gonzales	Sgt.
David Martin	Sgt.
Joseph Stanley	Cpl.
James E. Mayo	Cpl.
Elevinio Mendez	Cpl.
Rodney Cavell	Cpl.
Ferrell G. McKinley	Cpl.
Marcelino Serna	Cpl.

Rank

Name	Rank
Dane O. Carlton	Pfc.
Turner Lane Jr.	Pfc.
William A. Johnson	Pfc.
Frank Stefanik	Pfc.
Wendell L. Griffith	Pfc.
Irving Scheider	Pvt.
Harold L. Hart	Pvt.
William H. Stice	Pvt.
Orion K. Lambert	Pvt.
Jose Ascencion	Pvt.
Milton Frank Jr.	Pvt.
Richard J. Janisee	Pvt.
John B. Johnson	Pvt.
Robert W. Robinson	Pvt.
Harold L. Titwell	Pvt.
Tommie R. McDuffie	Pvt.
Roland E. Huffstutier .	Pvt.
Anton Andel	Pvt.
Richard B. Doby	Pvt.
Marshall Raines	Pvt.
Charles C. Bramble	Pvt.
Norman L. Christian	Pvt.
Herbert H. Beesley	Pvt.
Orin L. Floyd	Pvt.
Leonard L. Romonoski	Pvt.
Arthur M. Miller	Rct.
William W. Barritt	Rct.
Stefen Rapusinski	Rct.
Frencis Kerins	Ret.

7. Civilian Male Personnel

Name			Duty
14 cmic			1.7 CL G.V

Nicholas Flores	Clinic Technician
Charles Lozano	Ward Attendant
James T. Wynn	Ward Attendant
Charles Phillips	Ward Attendant
Archie R. Spears	Ward Attendant
Manuel Sainz	Ward Attendant
Erwin J. Pleasant	Ward Attendent

8. Civilian Female Personnel

Wiss	Anna Wilhe	elm Section	a Secretary	1	January-6	Aug.	1949
Mrs.	Frances B	reaux Section	on Secretary	6	August- 3	l Dec.	1943
				1	January-3	l Dec.	1949

CLINICAL RESEARCH

1. Peptic Mcer

During the entire period of this report, a clinical study of the efficacy of the anion exchange resin—resinat— was completed. An article, based on this study, was written and compiled by the Chief, Gastroenterology Section and Major Charles J. Hornisher, who was a Resident on the Section in 1948. A control group of patients was studied using the customary conservative medical treatment for uncomplicated peptic ulcer. The resinat group received the same treatment as the control group with one exception. In place of a colloidal aluminum hydroxide ant—acid, they were given an anion exchange resin.

2. Laennec's Cirrhosis of the Liver

Eighteen months of a 24 month study of the effectiveness of the combined intensive therapy for portal or Laennec's cirrhosis of the liver was completed. We hope to publish our results sometime in 1950.

3. Post-vagotomy Survey

During the latter part of 1949 the Chief, Gastroenterology Section has been one member of a post-vagotomy survey study committee, which also includes Lt. Col. Jack Segal, Lt. Col. Glenn J. Collins, and headed by Col. Joseph H. Schaeffer, Chief, Surgical Service. In the past, about 60 vagotomies (most of which have been combined with other ulcer surgery) have been performed at the Brooke General Hospital.

The American Gastroenterological Association formed a committee to study the results in patients following surgical vagotomy. Certain forms were sent to selected hospitals over the country to be filled out after the follow-up studies on the patients.

At year's end we have surveyed about 20 of this group. This survey included the Hollander insulin test, histamine fractional gastric analysis, acid pain test, 12 hour overnight secretory test, gastroscopic examination, and gastro-intestinal roentgen series. All of this work was done on the Gastroenterology Section with the exception of the Hollander test.

4. Amebiasis

The Chief, Gastroenterology Section, is a member of a group headed by Col. Dwight Kuhns, Commanding Officer of the Fourth Ammy Area Medical Laboratory, to study the effectiveness of aureomycin in the treatment of amebiasis.

The patients are placed on a controlled dose of aureomycin and followed carefully with procto-sigmoidoscopy, frequent stool examinations, course and symptomatology.

5. Biliary Tract

The Chief, Gastroenterology Section, with collaboration by Major John M. Masen of the laboratory service, has been working since July 1343 on a new laboratory procedure for the more accurate differentiation of gall-bladder bile from liver bile recovered by diagnostic biliary drainage. This study may be completed in 1950.

V

CLINICAL DATA

1. Ward 25

Cases reported below are those discharged from hospital during the period of this report.

		Total	Cases	
Code Number	Diagnosis	1948	1949	Total
010-151	Amebiasis, chronic, mild	3	13	16
660-151	Amebic colitis	2	1	3
002-x34	Anorexia nervosa	1	0	1
670-887y	Anus, adenocarcinoma,			
	suspect	0	1	1
670-400.1	Anus, fissure of	1	1	2
661-1x0	Appendicitis, chronic	1	1 .	2
040-887.9	Carcinomatosis, generaliz	sed		
	abdominal	1	0	1
682-100	Cholangitis, chronic	1	0	1
687-100	Cholecystitis, acute	1	0	1
687-100.0	Cholecystitis, chronic	8	. 7	15

		Total Cases		
Code Number	Diagnosis	1948	1349	Total
687-100	Cholecystitis, chronic,			
	atrophic	0	1	1
687-615	Cholelithiasis	5	1	6
660-151	Colitis, chronic-due to			
	endameba histolytica	9	3	12
660-557	Colitis, mucous	4	1	5
660-951	Colitis, ulcerative	0	4	4
660-887	Colon, adenocarcinoma	0	2	2
660-642.0	Colon, diverticulosis	6	5	11
660-642.0	Colon, diverticulitis	0	1	1
660-580	Colon, irritable	4	0	4
604-y00.9	Diarrhea, cause unknown	6	0	6
651-036	Duodenum, diverticulum	3	7	10
651-190	Duodenitis, organism unknown	1	1	2
651-930	Duodenitis, subacute	0	1	1
660-116	Dysentery, bacillary	2	2	4
637-100	Esophagitis, due to infection	n 1	0	1
637-837	Esophagus, adenocarcinoma	0	1	1
63x-641	Esophagus, varix of	3	3	6
604-100	Enterocolitis, acute	1	0	1
640-550	Functional GI disturbance	10	1	11
640-100.0	Gastritis, acute	1	2	3
640-332	Gastritis, acute, due to			
	alcohol	11	8	19
640-383	Gastritis, acute, simple,			
	exogenous	6	24	30
640-000.0	Gastritis, acute, toxic,			
	ant poison	0	1	1
640-940.9	Gastritis, chronic, atrophic	1	1	2
640-940	Gastritis, chronic, nonspecif		2	6
640-940.6	Gastritis, chronic, hypertrop	_	2	2
640-147	Gastritis, syphilitic	1	0	1
6012-332	Gastroduodenitis, chronic,	_	_	_
	due to ethyl alcohol	1	2	3
6012-190	Gastroduodenitis, chronic,			- 4
003 000	recurrent, cause undetermine	d 14	0	14
601-930	Gastroenteritis, acute, cause	3.0	3.0	70
003 707	undetermined	12	18	30
601-383	Gastroenteritis, due to food		0	par.
003 100	poisoning-undetermined type	3	2	5
601–190	Gastroenteritis, due to	7	0	7
601-1141	infection	1	0	1
001-1141	Gastroenteritis, due to	1	0	3
	salmonella paratyph.	1	U	1

		Tot	al Cases	
Code Numb	er Diagnosis	1943	1949	Total
601-105	Gastroenteritis, due to	_		0
007 2202	staphlococcus toxin	0	2	2
601-1161	Gastroenteritis, chronic, due	0	1	1
003 070	to Shigella	0	1	1
601-930	Gastroenteritis, due to	2	7	E
603 800	unknown cause	2	3	5
601-390	Gastrointestinal allergy Giardia infestation-intestine	2	12	14
604-155 640-y00.7	Hematemesis, cause undetermined	5	3	8
651-951.7	Hemorrhage-duodenal ulcer	2	4	6
y00-600	Hemorrhage, GI tract, cause	~	•	0
300-000	undetermined	10	4	14
604-y00.7	Hemorrhage, intestinal, due to	10	-	TA
004-900.1	undetermined cause—probably			
	gastritis	3	0	3
60x-641	Hemorrhoids, external and			
00%-087	internal	4	1	5
66x-641	Hemorrhoids, internal	6	3	9
680-100.0	Hepatitis, acute, due to infection		4	9
680-332	Hepatitis, due to ethyl alcohol,		•	
	chronic, mild	1	0	1
680-151	Hepatitis, subacute, due to			
	endameba histolytica	0	1	1
680-100.0	Hepatitis, chronic, due to			_
	infection	1	0	1
275-100	Hernia, diaphragmatic, cause		,	
	undetermined	0	2	2
275-037.9	Hernia, esophageal hiatus,			
	congenital, mild	1	1	2
651-023	Herniation of gastric mucosa			
	into duodenum	1	0	1
600-x10	Idiopathic steatorrhea	1	0	1
640-x32	Indigestion	0	3	3
640-x33	Indigestion, chronic, gastric	0	2	2
604-y00.7	Intestinal hemorrhage due to			
	undetermined cause	3	1	4
650-415.4	Intestinal obstruction, due to			
	post-operative adhesion	0	1	1
604-y00.4	Intestinal obstruction, cause		_	
070 0010	undetermined	0	1	1
650-2612	Intestine, small, infestation,	0		
000 153	T. Saginata	0	1	1
660–151	Intestinal parasitosis, due to			
	endameba histolytica, chronic,	7	0	1
	moderately severe, improved	1	0	1

			Total Cas	8
Code Number	Diagnosis	1948	1949	Total
680-100.2	Liver, abscess, amebic	0	1	1
680-887.9	Liver, adenocarcinoma, secondary	0	2	2
6680-753	Liver, fatty, associated with			
	obesity	0	1	1
680-956	Liver, Laennec's cirrhosis	11	32	43
680-yx6	Liver, hepatomegaly, undetermin-			
	ed origin	3	0	3
014-711	Malnutrition in an adult person	,		
	caused by starvation	0	1	1
604-244	Necator Americanus (Ankylo-			
	stomiasis)	18	15	33
660-y00	No disease- amebiasis suspect	3	7	10
650-001	No disease- observation for			
	intestinal parasites	2	1	3
600-000	No disease found- observation			
	for GI disturbance	94	151	245
651 -y 00	No disease-observation for	_		
003 003	peptic ulcer	7	0	7
661-001	Observation- appendicitis,		~	-
00400 B	no disease	2	3	5
604-y00.7	Observation for intestinal	^	3	-
60700	hemorrhage-no disease	0	1	1
687 -y 00	Observation-cholecystitis, no disease	1	0	- 1
651-951.4	Obstruction-intestinal, due	T	0	T
001-201-4	to ulcer	0	1	1
690-1x0	Pancreatitis, chronic, cause	0	-	-
030-120	undetermined, improved	1	0	1
690-190.7	Pancreatitis, acute, severe,			-
000-20041	hemorrhagic	1	0	1
060-100	Peritonitis, acute, due to	Alle		-
000 200	unknown cause	1	0	1
668-944	Polyp- rectum, cause unknown	2	3	5
604-886	Polyposis, adenomatous, intestina	10	2	2
604-002	Post-vegotomy follow-up survey			
	study	0	20	20
668-100	Proctitis, acute	1	2	3
668-151	Proctitis, amebic	0	1	1
668-100.6	Proctitis, chronic	2	2	4
600-550	Psychogenic GI reaction	3	13	16
645-954	Pylorus- stenosis of	0	1	1
666-190	Rectosigmoiditis, chronic, mild	0	1	1
668-887	Rectum- adenocarcinoma of	0	1	1
640-887	Stomach- adenocarcinoma of	2	4	6

			Total Cas	es
Code Number	Diagnosis	1948	1949	Total
640-951.3	Stomach-ulcer, perforation of	f 1	1	2
640-951	Stomach-peptic ulcer of	6	10	16
604-256	Strongyloidiasis	1	1	2
660-254	Trichuriasis-colon	2	2	4
604-1541	Trichomonous infection, small	.1		
	intestine	0	1	1
640-951.7	Ulcer, peptic, of stomach,			
	multiple, with hemorrhage	5	1	6
651-951	Ulcer, peptic, duodenum	118	172	290
651-951.3	Ulcer, duodenal, perforation	10	3	13
650-951.4	Ulcer, peptic, duodenal, with			
	obstruction, chronic	1	0	1
600-y00	Undiagnosed. Observation			
	for GI disease	1	1	2

2. Consultations

With the exception of Ward 25, a 55 bed ward for enlisted personnel and veteran patients, the Gastroenterology Section serves the Brooke General Hospital on a consultation basis conducted by the Chief of Section. During the period of this report, about 1000 patients have been seen in consultation.

3. Gastroenterology Section Clinic

A- E	Procto-sigmoidoscopy	1948	1949	Total
8	a. Total male patients	- 273	264	537
t	. Male patients, military	- 138	99	237
0	. Male patients, civilian	- 7	19	26
d	d. Male patients, VA	- 128	146	274
€	e. Total female patients	- 49	36	85
1	f. Female patients, military	- 6	4	10
8	g. Female patients, civilian	- 41	31	72
k	n. Female patients, VA	_ 2	1	3
1	. Total patients	- 322	300	622
1	. Diagnoses			
	1) Normal	64	80	144
	2) Incidence of disease			
	Anal fissure	- 10	16	26
	Anal proctitis	- 80	77	157
	Anal fistula		0	1
	Anal polyp	- 0	5	5
	Amebiasis —	_ 2	8	10

Incidence of Disease cont'd.	1948	1949	Total
Amebic procto-sigmoiditis	12	0	12
Allergic recto-sigmoiditis		1	2
Angulation, marked, colon		1	2
Atresia, rectum		1	5
Atresia-sigmoid colon		1	1
Anal papillae-hypertrophied		15	18
Atonic anal sphincter		4	6
Anal cryptitis		13	15
Carcinoma-rectum	1	3	4
Colitis,ulcerative,chronic	10	14	24
Erosive anal proctitis	4	10	14
Giardiasis	0	1	1
Hemorrhoids, external	50	11	61
Hemorrhoids, internal	107	45	152
Hypertonic anal sphincter	36	22	58
Hypotonic anal sphincter	2	1	3
Mass, recto-sigmoid junction,			
polypoid -	0	1	1
Mucous colitis	2	1	3
Peri-anal skin disease		7	14
Proctitis, ulcerative	0	1	1
Prostatic disease		9	22
Redundant anal mucosa		22	36
Redundant recto-sigmoid mucosa		4	7
Rectal polyp		8	18
Recto-sigmoiditis		13	54
Recto-sigmoiditis, amebic	0	2	2
Recto-sigmoiditis, granular,			
ulcerative		1	1
Polyposis of sigmoid	1	1	2
Pseudo-polyposis-colon	2	0	2
Spastic colon	51	21	72
Skin tags, anal	13	19	32
Trichuriasis	0	1	1
B. Fractional Gastric Analysis	1948	1949	Total
Control of the Contro			
a. Total male patients		108	243
b. Male patients, military		48	103
c. Male patients, civilian		2	4
d. Male patients, VA		58	136
e. Total female patients		8	18
f. Female patients, military		1	4
g. Female patients, civilian	- 7	7	14

1948	1949	Total
h. Female patients, VA ———————————————————————————————————	0 116	0 261
j. Diagnoses 1) Normal ———— 62 2) Organic disease impression	36	98
Apparent achlorhydria - 2	3	5
Chronic gastritis —— 0	5	5
Erosive gastritis —— 7 Hyperacid gastritis —— 6	5	12
Subacid gastritis ———————————————————————————————————	0 2	6 3
Esophagitis — 3	Õ	3
Gastric hyperacidity - 51	56	107
Gastric hypersecretion- 18	12	30
Gastric hypoacidity —— 22	13	35
Gastric hyposecretion— 0 12 hour gastric retention 7	2	2
3) Functional diseases	6.	9
Gastric hypermotility - 34	12	46
Gastric hypomotility — 7	0	7
Pylorospasm4	3	7
C. <u>Diagnostic Biliary Drainage</u> 1948	1949	Total
	1949 86	249
a. Total male patients ————————————————————————————————————	86	249 107
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1	86 33 0	249 107 1
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88	86 33 0 56	249 107 1 144
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44	86 33 0 56 24	249 107 1 144 68
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8	86 33 0 56	249 107 1 144
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36	86 33 0 56 24 5	249 107 1 144 68
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207	86 33 0 56 24 5	249 107 1 144 68 13 53
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses	86 33 0 56 24 5 17 2	249 107 1 144 68 13 53 2 317
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207	86 33 0 56 24 5 17	249 107 1 144 68 13 53 2
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Diagnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22	86 33 0 56 24 5 17 2	249 107 1 144 68 13 53 2 317
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22 Cholangitis — 26	86 33 0 56 24 5 17 2 110	249 107 1 144 68 13 53 2 317
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22 Cholangitis — 26 Choledochitis — 25	86 33 0 56 24 5 17 2 110	249 107 1 144 68 13 53 2 317 94
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22 Cholangitis — 26 Choledochitis — 25 Cholecystitis — 6	86 33 0 56 24 5 17 2 110 39	249 107 1 144 68 13 53 2 317 94
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22 Cholangitis — 26 Choledochitis — 25 Cholecystitis — 6 Cholelithiasis — 7	86 33 0 56 24 5 17 2 110 39	249 107 1 144 68 13 53 2 317 94 38 33 25 11
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22 Cholangitis — 25 Choledochitis — 25 Cholecystitis — 6 Cholecystitis, early — 3	86 33 0 56 24 5 17 2 110 39	249 107 1 144 68 13 53 2 317 94 38 33 25 11 10 3
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22 Cholangitis — 26 Choledochitis — 25 Cholecystitis — 6 Cholecystitis, early — 3	86 33 0 56 24 5 17 2 110 39	249 107 1 144 68 13 53 2 317 94 38 33 25 11
a. Total male patients — 163 b. Male patients, military — 74 c. Male patients, civilian — 1 d. Male patients, VA — 88 e. Total female patients — 44 f. Female patients, military — 8 g. Female patients, civilian — 36 h. Female patients, VA — 0 i. Total patients — 207 j. Disgnoses 1) Normal — 55 2) Disease Impressions Bile tract stasis — 22 Cholangitis — 26 Choledochitis — 25 Cholecystitis — 6 Cholecystitis, early — 3 Chronic gastritis — 0	86 33 0 56 24 5 17 2 110 39	249 107 1 144 68 13 53 2 317 94 38 33 25 11 10 3

<u>1</u>	948	1949	Total
Gallbladder stasis ———————————————————————————————————		24 1	75 4
(gallbladder present) Strongyloidiasis Total obstruction-sphincter	1	9	34 2
of Oddi		0	2
Atonic gall-bladder	1	9	7
Failure to pass pylorus —		2 3	3 8
Dyskinesia, sphincter of			0
Oddi	27	16	43
D. Gastroscopy 1	948	1949	Total
a. Total male patients	51	108	159
b. Male patients, military	21	36	57
c. Male patients, civilian	1	5	6
d. Male patients, VA	29	67	96
e. Total female patients	2	15	17
f. Female patients, military		1	1
g. Female patients, civilian -	2	14	16
h. Female patients, VA	0	0	0
i. Total patients j. <u>Diagnoses</u>	53	123	176
1) Normal -	30	50	80
2) Disease found			
Carcinoma	2	5	7
Deformity of angulus	2	9	11
Erosions	0	2	2
Contracted gastro-enter-			
ostomy stoma	0	1	1
Atrophic mucosa	0	1	1
Atrophic gastritis	1	0	1
Failure to pass scope — Gastrospasm ————	0	3 4	3 5
Gastric ulcer-benign	8	10	18
Hypertrophied rugae	4	10	14
Hypertrophic gastritis -	1	2	3
Gastric diverticulum —	2	0	2
Polyp	0	2	2
Mucosa, gastric, friable-	0	1	1
Mucosa, pocketing of	0	1	1

	1948	1949	Total
Redundancy, antral mucosa	- 0	2	2
Sphincter, pyloric, hypotonic-		ĩ	ĩ
Pseudo-diverticulum	- 0	2	2
Pylorospasm	- 0	3	3
Pressure defect-prepyloric			
region	- 0	1	1
Pylorus, possible obstruction-	- 0	-1	1
Stomal gastritis	- 0	2	2
Superficial gastritis	- 5	18	23

4. Case Abstracts

The following abstracts of cases, diagnosed and treated on Ward 25, Gastroenterology Section, have been useful in the teaching of Residents.

Case 1.

P.F., a 23 year old white male was admitted for interval appendectomy with the history of previous admission in February 1948 for appendiceal abscess that had been treated conservatively. On August 20th 1948 appendectomy was performed and a normal appendix that was kinked and bound down by adhesions was removed. Postoperative course was characterized by low grade afternoon fever with recurrent nausea, vomiting and headache. His history revealed nausea, vomiting and headaches since 1945. He was transferred from surgery to medicine. Ward 25. for further diagnosis. Gastro-intestinal allergy was suspicioned. Physical examination showed boggy, gray turbinates, otherwise negative. The patient was placed on an elimination diet. On this diet, the patient's symptoms of nausea, vomiting, and headache rapidly disappeared. At the time of discharge from hospital, he was making experimental additions to the diet and was asymptomatic. Diagnosis- Apparent gastro-intestinal allergy.

Case 2

P.G., a 31 year old Latin-American male, was admitted on 5 August 1948 with diarrhea and weight loss. The patient's family history was noncontributory. His past history was negative except an attack of double pneumonia at eleven and 'rheumatism' at nineteen.

In the summer of 1944, while on manouvers in Texas, he developed diarrhea associated with cramping abdominal pain.

The symptoms lasted about a week and he received symptomatic treatment from the dispensary. The onset was stormy, and he passed six to eight large loose stools a day. The diarrhea subsided spontaneously. The last episode of diarrhea was in June 1948. Since then the patient lost 30 pounds in weight. He passes six stools a day and they are light brown in color, bulky, malodorous, without blood nor mucus. Physical examination on admission showed a malnourished Latin-American of average intelligence, 69 inches tall, weight- 130 pounds, skin clear, tongue slightly reddened at the edges. Otherwise negative.

A stool specimen passed on admission at the laboratory was "too oily for satisfactory examination". This oil stained as neutral fat. On this and subsequent stool examinations, no parasites, ova, nor blood were found. Culture for amebae was negative. Blood count, blood calcium, carbohydrate tolerance were essentially normal. A 24 hour stool weighed 670 gm., total solids 14.3 per cent, total fat (wet) 6.3 per cent, of this, fatty acid was 85 per cent, neutral fat 15 per cent.

Analysis of duodenal content for pancreatic enzymes was: amylase, 86 units, lipase 1.6cc N/20 NaOH/ml, trypsin-none, acid phosphatase- 0.8 Bodansky units, alkaline phosphatase- 13.8 Bodansky units, phosphorus- 1.7 mg.per cent. These values were confirmed by a second analysis in which the amylase was 272 units, lipase- 2.5cc, trypsin-none. Liver function tests and serum protein were normal.

X-ray of the chemt was normal. Cholecystography was normal. Barium enema was normal. The duodenum and small intestine showed complete loss of the normal mucosal pattern. There were numerous small filling defects within the small bowel. A number of loops were narrowed and irregular in outline. Barium tended to flocculate within the lumen of the small bowel.

During the first week the patient received no treatment other than a bland diet. No improvement followed. He was then placed on a low fat diet and the stools quickly became formed and the patient began to gain weight. A second cholecystographic examination revealed many small stones in the gall-bladder. The patient was operated and a cholecystectomy performed. The gall-bladder was found to be full of small gall-stones.

Case 3

J. S., a 52 year old, white male, was admitted on 25 August 1948 complaining of diarrhea for the past two and a half days. His family history and personal past history were noncontributory. On 22 August, the patient ate some food that they were suspicious of. Both the patient and his wife began to have diarrhea about two o'clock the next morning and it persisted until the time of admission to the hospital. They both ran a fever. On 25 August

the patient's stools became thicker and tarry. Physical examination on admission was negative with the exception of a temperature of 101.4 F., WBC-5,000 with 80 percent lymphocytes, 9 eosinophiles, and 10 neutrophiles. The next day the white count was 5600 with 66 per cent neutrophiles and 34 percent lymphocytes. The hematocrit was 47, Hb.-15.8 gms. Plasma proteins-7.45gm. On August 27th his white count was 5400 with 60 percent neutrophiles, 36 per cent lymphocytes, two per cent monocytes, and two per cent eosinophiles. A rectal swab was done on admission and a culture of this swab showed salmonella. Immediately after taking the culture, the patient was started on sulfadiazine with a prompt febrile and clinical remission of all his symptoms.

Diagnosis: Gastroenteritis, acute, severe, due to Salmonella.

Case 4

C.G., a 45 year old white male, was admitted to the hospital 6 May 1948 complaining of constipation and fullness of the abdomen. His present illness began about a year before with excess gas and indigestion for which he received a series of colonic irrigations.

For the last month prior to admission he had anorexia. Three days before admission he developed nausea and vomiting. During that time he had been receiving sulfa drugs. His diet was unusual in that it consisted mostly of peas and corn bread. He rarely ate meat. He had a history of 'moderate' alcoholism. His family history and personal past history were noncontributory.

Physical examination on admission revealed the abdomen to be tensely distended with fluid but no palpable masses. Rectal examination showed three external skin tags. The prostate felt normal. There were telangiectases on the anterior chest and abdomen. He showed palmar erythema and the hair was scanty in the armpits and no hair was present on the chest. The liver was palpated three fingers below the costal margin. The patient stated that he had had a questionable jaundice two years before admission. He had pitting edema of both lower legs, and a rather loud systolic murmur at the apex.

Laboratory: Profound anemia-8.0 gm. Hb., WBC normal. Urinalysis showed a rare granular cast, specific gravity-1.026, STS negative. Bromsulfalein test-29 percent of dye remaining after 45 minutes. Total plasma protein-6.3 gm. A/G ratio-1.0. Prothrombin time was 26 seconds. Icterus index

4.3mg. per cent, Cephalin cholesterol flocculation- two plus after 24 hours and two plus after 48 hours. Thymol turbidity test- 13.5 units.

Abdominal paracentesis was performed on 13 May and over 5000 cc. of amber fluid was removed. Cellular study revealed no evidence of malignancy. The patient was placed on a high protein diet and was given several plasma and blood transfusions. His abdomen again filled with fluid and a second paracentesis was performed on 25 May. Since then, to date, the patient has not had a return of excess fluid in the abdomen.

From 12 May until 24 July 1948, the patient was on a modified Patek regime of medical treatment for Laennec's cirrhosis. This consisted of a high carbohydrate, high protein, low fat diet with vitamin B. complex by mouth and two cc. of liver extract intramuscularly daily. On this regime he had no recurrence of ascites, his BSP fell to a plateau of about 12 per cent remaining, but his thymol turbidity test gradually rose. His cephalin cholesterol flocculation test dropped to zero at 24 hours and one plus at 43 hours.

on 21 July 1948 a needle biopsy of the liver was performed and a diagnosis made of Laennec's cirrhosis of the liver. The patient was changed to the combined intensive regime of treatment. This consisted of a high protein (three meat servings daily, six to eight glasses of skinmed milk, and cottage cheese with evening mess), high carbohydrate, moderate fat diet. Vitamin B complex (potent) with intraheptol were given intravenously daily, vitamin B complex and potent total vitamins by mouth, and protolysate by mouth. The total caloric intake was about 3000 daily. The patient rapidly improved and at the end of three months was discharged to normal activity. His BSP had dropped to eight per cent remaining dye and the thymol turbidity had lowered to 26 units. The patient was free of all signs and symptoms at the end of three weeks on the combined intensive therapy regime.

At this writing, he has been on normal activity for 14 months with no recurrence of ascites or symptoms.

Case 5

R.K., a 19 year old white male was admitted to the hospital on 13 July 1943. Hischief complaints were abdominal pain and diarrhea. He had been well until noon of that day when he had the sudden onset of epigastric pain immediately after eating. He had a sense of abdominal fullness. Later he developed diarrhea with brown watery fleces which, on one occasion, contained blood.

Physical examination showed a well-developed, well-nourished, white young male with a temperature of 103° F. There was some tenderness on palpation in the left upper quadrant of the abdomen.

Laboratory: WBC- 4,300, 66 per cent neutrophiles, 30 percent lymphocytes, 4 percent monocytes. Blood smears were negative for malaria. Feces were negative for parasites or occult blood. A culture of the stool demonstrated Shigella paradysenteriae Sonei strain. The patient was placed on sulfadiazine immediately after culture. His stools became solid on 17 July and on that same day he became afebrile. Sulfadiazine was discontinued on 19 July 1943. His stools at that time and since were negative for pathogenic organisms.

Case 6

L.C., a 39 year old white male, was admitted to the hospital on 3 June 1948 complaining of recurrent fever and abrupt loss of weight. The patient's past history revealed that four years before he had a severe diarrhea of three weeks duration. In January 1946 he had a bout of right upper quadrant abdominal pain which persisted four days and was relieved by taking castor oil. In August 1946, he had a severe bout of chills and fever, which was diagnosed as malaria and treated with atebrine. In April 1947 he had another bout of chills and fever and was again treated with atabrine. In April 1948 he again developed chills and fever. Ten malaria smears at the hospital here were negative for malaria. He was released without treatment at his own request and during the next two months lost 50 pounds in weight. He had episodes of chills and fever but no vomiting nor diarrhea. He noticed one black stool three weeks before admission. A local physician had a gastro-intestinal x-ray series performed and found a duodenal ulcer. He was admitted to Brooke General Hospital on 3 June 1948.

Physical examination showed an acutely ill white man appearing older than his stated age. His skin was pallid and he was perspiring profusely. He weighed 150 pounds (his usual weight-200-210 pounds). Blood pressure- 100/70. There was tenderness on deep palpation in the right upper quadrant of the abdomen.

Laboratory: RBC- 3.7 million, Hb.-12.0 gm., hematocrit-31, WBC-16,300 with 88 percent neutrophiles and 12 percent lymphocytes. The feces were negative for parasites or occult blood. Serological test for lues and for the common agglutinins were negative. Weil-Felix was negative. Blood cultures were negative on two occasions. On 25 June, examination of feces found cysts of endameba histolytica. This was found again in the next day's stool. A gastro-intestinal x-ray series showed a constant deformity of the duodenal bulb.

The patient ran a continuous fever from the date of admission until 10 Jung, occasionally to 1030 F. He complained of severe chills and fever with drenching sweats. On 10 June he was given aralen, although smears for malaria had been repeatedly negative. Response to this drug was abrupt and dramatic. Thereafter, he had no fever. The patient gained weight rapidly on a convalescent ulcer regime including resinat. Because of the cysts of endameba histolytica, he was given a course of emetine, diodoquin, and carbasone. Following this. repeated examinations of the stool with and without purgation. revealed no cysts nor pathogens of other sort. Gastro-intestinal x-ray series on 6 July was entirely negative. The patient gained about 40 pounds during hospitalization. At discharge from the hospital on 21 July 1948, he was asymptomatic.

Diagnosis: 1. Ulcer, peptic, duodenum, chronic.

2. Colitis, amebic, due to endameba histolytica.

Case 7

C.H., a 22 year old white male, was admitted to the hospital on 21 June 1948 complaining of epigastric fullness of one week duration. The patient's family history and personal past history were not contributory. He stated that his distress was insidious in onset, that he had frequent episodes of a similar nature during the past three years. The first attack followed a period of diarrhea while stationed in Guan.

The present episode began one week before admission and became progressively severe with a loss of appetite and a loss of 16 pounds in weight. He was nauseated and vomited twice. The vomitus was greenish-yellow. There was no diarrhea, but slight constipation. The stools were dark green to black in color. At the beginning his stools were almost white in color. His urine had been dark for the past week. Other people told him that his eyes were yellow.

Physical examination on admission showed a well-developed

well-nourished, intelligent young man. He was 70 inches tall and weighed 142 pounds. His sclerae were slightly icteric. The liver and spleen were not palpable.

Laboratory: Icterus index-32 mg.%, Van den Berghdirect 4.6, prothrombin time 29 seconds with a control of 21, cephalin cholesterol flocculation was 2 plus at 24 hours and 3 plus at 48 hours. Thymol turbidity was 28 units. The feces were positive for urobilin.

The patient was placed on a high carbohydrate, high protein, low fat diet. His clinical improvement was excellent. By 3 July his icterus index was 3. On 21 July the BSP showed 0 percent of dye remaining, and the thymol turbidity was 9.8 units. The patient was asymptomatic.

Diagnosis: Hepatitis, acute, due to infection.

Case 8

W.W., a 60 year old white male was admitted on 19
December 1947. He was in excellent health two months
before admission. At that time he noticed the onset of
slight nausea. This seemed to be related to the taking
of food and was relieved by lying down. He was treated
symptomatically by a local physician but did not
improve. His nausea became more pronounced following meals,
but he had no vomiting. One evening the patient noticed
a mass in the abdomen that pulsated. He was sent to the
Brooke General Hospital.

Physical examination revealed an elderly white male fisherman, whose skin was rough and keratotic. Blood pressure—right arm, 118/68; left arm, 110/60; right leg, 0/0; and left leg, 98/60. Abdominal examination revealed moderate tenderness in the left upper quadrant on deep palpation. There was a pulsatile expansile mass about the size of an orange in the left para-umbilical region.

The patient was asymptomatic and was placed on a smooth diet. On 2 January 1948, an audible bruit was heard over the pulsatile mass. X-ray examination with a flat plate of the abdomen: "There was no evidence of intestinal obstruction. There is a questionable shadow along the left side of the spine at the level of L-3 and L-4, and a faint plaque of increased density which may represent calcification of the wall of the abdominal aorta". On 22 December a barium enema reported: There is no evidence of intrinsic disease of the colon. The palpable pulsating mass just to the left of the mid abdomen displaces a portion of the

transverse colon anteriorly, but is entirely separate from it". On 23 December a gastro-intestinal x-ray series reported: "Esophagus, stomach, and duodenum show no intrinsic abnormality. The inferior portion of the fundus of the stomach is displaced forward by the pulsating mass. The mass previously described is believed to represent an aneurysm of the abdominal aorta. It is centered on L-3 and L-4 and the calcification previously described is again demonstrated". Diagnosis: Aneurysm, abdominal aorta, unimproved, cause undetermined.

Case 9

E.B., an 18 year old white male, was admitted to Brooke General Hospital 1 September with chief complaint of intermittent pains in the abdomen of four days duration. On 27 August 1949, the patient developed pains in his lower abdomen which were intermittent, but persistent until the day of admission. He also had moderate anorexia but no vomiting nor diarrhea. On 31 August he was seen in the Emergency Room, Brooke General Hospital, where it was suspected he had appendicitis. After a short period of observation, he was transferred to the gastroenterology section for further diagnosis.

The patient's past history was noncontributory as was his family history. Upon admission to Ward 25 the patient appeared to be well developed, well-nourished, white male of stated age, not acutely ill. His temperature was 100° p and blood pressure-105/70. All of his physical examination was within normal limits except for slight tenderness in the right upper quadrant and a palpable liver which descended three fingerbreadths below the right costal margin on deep inspiration. The liver edge was sharp and of normal consistency. There was only slight tenderness to fist percussion in this region. No masses nor other organs were palpable in the abdomen.

All of his laboratory work was normal with the exception of a sedimentation rate of 18mm in one hour. Stool examinations revealed the ova of Trichuria trichura and hookworm.

The patient was given 15 grains of hexylresorcinol crystals (crystoids) and the usual follow-up treatment. During his stay on Ward 25, he remained completely asymptomatic. He was discharged as cured on 14 September 1949.

Diagnosis: 1. Trichuris trichiura of the colon.

2. Ancylostomiasis, intestinal.

C.B., a 48 year old white male, was admitted to Brooke General Hospital on 5 May 1943 with the chief complaints of epigastric and left upper quadrant pain and occasional morning vomiting. The patient associated the onset of the occasional episodes of epigastric distress with nervous excitement occurring in July 1918. He was treated by his father. who was a physician, and after about two and a half years felt perfectly well until 1937 when his epigastric distress recurred with greater severity. His pain was present almost daily and usually recurred in the afternoon. He obtained relief after vomiting and on taking baking soda. His symptoms continued to become more severe until December 1942 when he had a very sudden seizure of pain in the epigastrium while walking in the street. This caused him to fall to the ground and he vomited profusely. This was accompanied by profuse sweating and he felt extremely weak. The pain at that time was localized in the epigastric region. He was hospitalized that same day and was operated upon as an emergency for perforation of the intestine. He was told that he had an ulcer of the duodenum. After discharge from the hospital, he continued to have mild to moderate epigastric pain in spite of an ulcer diet and amphogel. From that time until now he has had recurrent attacks of pain in the epigastrium occurring usually in the afternoon, but during the past three months, it has become more constant and severe. He has vomited about once a week during the last three months but has seen no blood.

Physical examination on admission to Brooke General Hospital revealed a well healed scar running from just to the right of the xyphoid to a point one and a half cm. above the umbilicus. It was soft and no enlarged organs or masses were palpated in the abdomen. His stools were positive for occult blood. Gastroscopic examination revealed a superficial gastritis. Gastro-intestinal x-ray series was suspicious for an ulcer on the greater curvature of the stomach. However, gastroscopy failed to confirm this, and also x-ray re-examination failed to confirm it.

The patient was placed on a convalescent ulcer diet, amphojel. Within 15 days, he was asymptomatic. He was subsequently placed on mucotin tablets and discharged from the hospital on 23 May 1949.

Diagnosis: Gastritis, chronic, moderate, cause undetermined, improved.

This case is presented as a proven case of gastritis by gastroscopy which mimicked the symptoms of peptic ulcer.

M.B., a 46 year old white male, was admitted to Brooke General Hospital on 12 July 1949 with the chief complaints of mild pain in the left chest, lasting for six to ten minutes. for one month prior to admission. This caused the patient to get an EKG, and he was subsequently admitted to the cardiac ward for study. At that time he had been leaning against a desk for approximately one hour and on straightening up noticed a pressure-like pain deep anteriorly at the left costal margin. The pain did not radiate and disappeared in about two hours. He had no recurrence of this pain until two days later, while mowing the lawn, the pain became severe. After this time he noticed that the pain appeared after mild exertion and was relieved by sitting or lying down. He also had several episodes of fluttering feeling at the left costal margin and under the 10th left rib in the mid-clavicular line. He also noticed extreme shortness of breath after climbing stair or walking a block and 'got his breath better' if he folded his pillow at night. The patient had no episodes of paroxysmal nocturnal edema. His pain was caused by eating fast, eating beans or fried foods, and he never had pain before breakfast but most always after breakfast. This exacerbation of pain was followed by remission until approximately three hours later or one hour before lunch. Lunch relieved the pain entirely. The pain did not radiate. An EKG was taken prior to admission to hospital and he was given advice to reduce in weight. One week prior to admission he noticed black tarry stools.

Physical examination was negative except for a feeling of fullness in the epigastrium but no definite mass and deep pressure over this area elicited the same type pain experienced by the patient

Laboratory: The stools were positive for cysts and trophozoites of giardia lamblia. Urea nitrogen-16.5 mg., sedimentation rate was 12 mm in one hour. Cholecystographic examination was negative. GI series showed a small diverticulum in the third part of the duodenum. The EKG in hospital showed Q waves in leads II and III which could be residuals of old posterior wall infarction. The patient remained on the cardiac ward until 19 July 1949 when he was transfered to Ward 25. While on the cardiac ward, studies failed to reveal any evidence of cardiovascular disease. He was transfered because of his left upper quadrant pain.

The patient was given a course of atabrine and stools promptly became negative for giardia by 28 July. At this time, the patient was asymptomatic and was discharged from hospital.

Diagnosis: Giardia infection of intestine, cured.

For many years, giardiasis was felt to be entirely non-pathogenic. This has not been the author's experience. This case demonstrates a patient with a large infestation of giardia accompanied by vague epigastric symptoms. It seems that this patient had intestinal involvement from the giardia which accounted for the melena and his pain.

Case 12

F.T., a 54 year old white male, entered the Brooke General Hospital on 15 November 1948 complaining of gas in the stomach. The patient had undergone an operation in February 1947 at which time a "gas tumor" was removed and a partial gastrectomy was done. His complaints then were right upper quadrant pain and gastric hemorrhage and weight loss of ten pounds. Melena had preceded the gastric hemorrhage. Following operation, the patient felt very well until three months prior to his last admission. At that time he noticed a pressure sensation in the epigastrium. This increased to the point where the least amount of fluid caused pressure in the upper abdomen. He had no nausea or vomiting except one episode five days prior to admission. He had been constipated for the last three months.

Physical examination on admission revealed a chronically ill appearing male with evidence of weight loss. The skin was loose with scattered benign nevi. He demonstrated peripheral vessel arteriosclerosis. There was a well healed scar in the enigastrium underneath which was felt an irregular nodule. The mass was six by eight cm., non-tender, firm, and moved with respiration. It was felt in the left upper quadrant and seemed to be attached to a mass in the left side of the epigastrium. Examination of Blumer's shelf revealed the same nodularity. The inguinal nodes bilaterally were firm and non-tender. Positive laboratory findings were: RBC- 3.5 million. WBC-13.800, Hb.9.5 gm... hematocrit- 31 VPC. Urinalysis showed a few RBC per HPF. Serological examination showed skin to be positive with quantitative Kahn of 10 units. Wasserman positive, and cardiolipin complement fixation tests positive. GI series 19 November 1948 revealed a large defect on the lesser curvature of the stomach proximal to the gastroenterostomy. There was a hard irregular mass palpable at the site of the mass below the left costal margin. The liver was seen to be enlarged. X-ray findings were consistent with recurrent carcinoma of the stomach and suggestive of metastases to the liver, which accounted for its enlargement. Gastroscopic examination gave the impression of probable recurrent areas of carcinoma with a large amount of

free fluid present.

On admission to the hospital, the patient was given a soft diet and supportive therapy. He was given various transfusions and medications but seemed to have a down hill course. He was seen in Surgery and no further surgical procedures indicated. On 23 December 1948 the patient began complaining of dull acheing in the region of the liver. He was continued on demerol and blood transfusions. The patient expired on 27 December 1948.

Diagnosis: (Clinical): Adenocarcinoma of the stomach with generalized carcinomatosis.

Summary of Autopsy:

At autopsy, a diffuse carcinomatosis was encountered. The abdominal cavity contained 1200 cc. of straw colored fluid. There was an old gastroduodenostomy and anterior gastrojejunostomy. The stomach wall was markedly thickened and composed of firm gray tissue. The mucosa was almost entirely ulcerated and on the surface there was a bile stained necrotic material. The thickness extended into the esophagus and jejunum. The microscopic picture was that of infiltrating carcinoma. Metastasis was present in the liver, spleen, lung, bladder, pleura and peritoneal surfaces. The kidneys showed slight arterio-nephro-sclerosis, and the arteries were generally sclerotic. Positive Wasserman and Kahn were found but chronic diagnosis of syphilis was not observed.

Case 13

G.B., a 43 year old white male, was admitted to Brooke General Hospital as a transfer from an Air Force Station Hospital on 28 January 1949 complaining of diarrhea.

His present illness revealed that he was discharged from the Army in August 1945 for nervousness. He was out of the Army for three months, during which time he gained 20 pounds in weight and on 8 November 1945 re-enlisted in the Army. In June 1947, while in the Azores, he again had diarrhea which became more frequent and contained excess mucus. He was sent to a Station Hospital where he was given a smooth diet, but in April 1948, while in New Foundland, he developed durther diarrhea. In May 1948 a diagnosis of chronic colitis was made. In December 1948 he was transferred to Brooke General Hospital.

Physical examination on admission showed an adult white male, six foot two inches tall, weighing 150 pounds. The patient was in no acute discomfort and appeared chronically ill. Routine

physical was negative except for very slight abdominal

splinting, generalized.

Laboratory: Sedimentation rate—38 mm. in one hour. Stool examinations revealed mucus, pus, and red blood cells. Proctosigmoidoscopic examination on 1 February 1949: "The proctosigmoidoscope was passed to the eight inch level where it was impossible to see. At that point fresh bleeding was encountered. Several nodular masses were seen which had a cartilaginous feel as the scope slipped over the nodules. Impression: Carcinoma of the rectum."

On 2 February, barium enema showed a constricting deformity about the recto-sigmoid junction. Clinical diagnosis was neoplastic mass in the rectosigmoid region of the bowel. The patient was transferred to

surgery.

On 18 February 1949 the patient was operated. On entering the peritoneal cavity, the liver was carefully explored and no evidence of metastasis was found. The sigmoid colon was identified. The pelvic sigmoid made an '0' type of turn and within this area was a large tumor mass which was fixed posteriorly and anterolaterally: two walls of the sigmoid were adherent to each other, and on attempting to break down this adherence, a small amount of pus exuded and appeared to be the presence of a perforating carcinoma. The retroperitoneal lymph node extending from the tumor upward above the bifurcation of the aorta appeared to be involved in carcinomatous growth. It was decided that the condition was inoperable. The wound was closed by bringing out a loop of the sigmoid colon for a permanent colostomy.

Case 14

M.C., a 37 year old Latin-American male, was admitted to Ward 25 from the emergency room, Brooke General Hospital on 6 June 1949 in a comatose state. A history was obtained from the patient's niece who revealed a heavy alcoholic history for the past three or four years. According to the niece, the present illness began nine days before admission when the patient began to vomit blood. One to two days before admission it was noticed that the patient

was jaundiced, that his abdomen was enlarged, and he had a bloody diarrhea. No contributory history could be obtained

of his family.

Physical examination made it obvious that the patient was in a morbid comatose state with generalized, moderate jaundice and numerous icteric areas about the extremities. There was dry blood about the mouth and gums. Moist rales were heard at the base of both lungs. The abdomen was moderately enlarged and a definite fluid wave could be demonstrated. One spider angiomata was detected on the right shoulder, posteriorly. The patient's right leg was missing below the knee.

Laboratory: WBC- 15,200- 87 percent polys.; Hb. 8.0 gms.,

icterus index-35 mg., blood urea nitrogen-30 mg.

Course in Hospital: On admission, the patient's temperature was 101°F. An emergency type and cross match of his blood was made, blood requested, and 1000 cc. of whole blood ordered. The patient was placed in an oxygen tent, and given Vitamin K intramuscularly. Because of thrashing about in bed and edema of the extremities, it was impossible to administer the whole blood. The patient deteriorated very rapidly and expired at 0600 hours on 7 June 1949.

Clinical Diagnosis: Laennec's cirrhosis of the liver and acute yellow atrophy.

Summary of Autopsy:

1. Myocardial hypertrophy.

2. Emphysema.

3. Congestion and edema both lungs, bilateral hydrothorax, partial atelectasis, both lower lobes.

4. Splenomegaly (450 gms.)

5. Moderate varices of the esophagus.

6. Ascites (2200 cc.)

7. Advanced Laennec's cirrhosis of the liver.

3. Acute cholecystitis with moderate cholelithiasis.

9. Slight bile nephrosis of the kidneys.

Case 15

E.C., a 27 year old white male was admitted to the Brooke General Hospital on 12 August 1949 with the chief complaint of diarrhea of one week duration. This was accompanied by headaches, fever, and tenesmus of one day duration. The onset of the patient's symptoms was about 8 August 1949 when he suffered a sudden onset of diarrhea and nausea without vomiting. During that day he passed approximately 15 loose watery bowel movements associated with some abdominal distention and cramping abdominal pains, localized mostly in the lower abdomen. On 10 August he developed almost continuous occipetal headaches and on 11 August he had fever for the first time, the temperature rising to a maximum of 102°F. On 12 August he was admitted to the dispensary and given a mixture of bismuth and paregoric and suffered a spell of syncope.

At the time of admission to the hospital, he complained mostly of weakness, fatigue, occipetal headache, and pain in the lower abdomen and left upper quadrant of the abdomen. The bowel movements were watery in consistency and contained flaky fecal material and threads of mucus. The patient had never been overseas, and his past history was non-contributory.

Physical examination revelaed a well-developed, well-nourished young white male who was 65 inches tall and weighed 174 pounds. Blood pressure- 134/84. There was generalized tenderness on palpation over the abdomen, especially in the lower half; otherwise normal.

Laboratory: Prior to his admission to the hospital, the patient sent one stool specimen to the Fourth Army Area Medical Laboratory which was reported positive for endameba histolytica by culture. Since his admission to the hospital the patient had four stool examinations, three of which showed trophozoites and cysts of endameba coli but negative for endameba histolytica. Urinalysis negative. Blood serology negative. Chest x-ray negative. Proctoscopic examination revelaed a few inactive external hemorrhoids and moderate granular recto-sigmoiditis with many pin point hemorrhagic ulcers in the sigmoid colon and one inch distal to the rectosigmoid junction. Direct smears from the bowel wall during proctosigmoidoscopy failed to reveal endameba histolytica.

Course in Hospital:

Because of the positive stool examination for endameba histolytica at the Fourth Army Area Medical Laboratory prior to admission, the patient was given course of diodoquin. Very shortly after starting medication, the bowel movements decreased to three to four per day and the stool became firm in consistency. On 23 August, stool culture was reported positive for Shigella sonnei, smooth. On 24 August the patient

was started on streptomycin, one gram every six hours, and continued on this medication through 29 August 1949. By that time the patient was asymptomatic and afebrile and he was returned to duty with instructions to continue the intake of diodoquin as an outpatient until he had 15 day's treatment.

Diagnosis: 1. Amebic proctitis, moderate, improved.

2. Dysentery, bacillary, Shigella variety, cured.

Case 16

W.C., a 32 year old white male, was admitted to the Brooke General Hospital for a second time after having signed out of the hospital against medical advice on 7 July 1949, with chief complaint of pain in the left arm. nervousness, nausea, and bloody vomitus. The patient had no trouble with his stomach until 1943 while in a prison camp in Wanchuria. He began with nausea and vomiting after every meal with cramping pain in the abdomen. He had episodes of similar attacks about once a month. During this time. he also had diarrhea with four or five bowel movements daily. These intermittent symptoms persisted until November 1945 after having been liberated from prison in August 1945. In July 1946, he was hospitalized because of chest pain, shortness of breath, and nervousness. The patient has been very nervous for the last five years with tremors of the hands. He has also complained of insomnia and restlessness during this time.

Physical examination was negative. All laboratory work was normal. History from a girl friend stated he had been drinking quite heavily. During his present hospital admission, he had no gastrointestinal complaints and he was thought to be a rather typical picture of chronic anxiety with the possibility of superimposed chronic alcoholism which may on occasions cause alcoholic gastritis. During his original admission, 16 June 1949, the patient was placed on a convalescent ulcer diet, phenobarbital, Vitamin B. This regime gave the patient slight relief but he remained very nervous and irritable. On 4 July the patient was given a pass because he stated he had some important marital matters to discuss with his wife. He did not return on 6 July when due. On 7 July the patient was returned to the ward from the emergency room where he had been treated for an icepick wound through the left shoulder that he had sustained during a fight with a Mexican in a downtown bar. He was extremely tense, irritable and very anxious to be discharged from the hospital in order to solve his domestic problems which he had not accomplished while on pass. The patient left again against medical advice.

Diagnosis: Psychogenic gastrointestinal reaction, chronic, moderate, unimproved.

Case 17

E.C., a 45 year old white male, was admitted to the hospital with no complaints. The patient was well until approximately one and a half years before when he suffered the onset of epigastric pain which was recurrent and intermittent. The distress was irregular but usually came three hours after meals and was relieved by ant-acids.

On 20 July 1949 he was admitted to another General Hospital with severe epigastric distress and rigid abdomen and was operated that day for perforated duodenal ulcer.

Physical examination on admission to Brooke General Hospital was negative. His laboratory work showed the chest x-ray negative; GI series showed a prominent inferior recess of the duodenal cap but no ulcer. His stool specimen on 15 and 18 August showed ova of trichuriasis. The patient was given a saline purgative followed by one gram of hexylresorcinol. Following this treatment, the stools were consistently negative for ova or parasites. Diagnosis: Trichuriasis of colon, cured.

Case 18

C.C., a 30 year old white male was admitted to the Brooke General Hospital on 2 June 1948 as a transfer from overseas. He gave a history of having had epigastric pain for three weeks prior to being admitted to a Station Hospital overseas. His pain and vomiting began about 30 minutes after each meal. He frequently noted coffee ground material in the vomitus. On the day he entered Brooke General Hospital there was a small amount of bright red blood with the vomitus followed by syncope. He had had tarry stools for two or three days prior to this episode. He was placed on a modified Sippy diet and he gradually improved.

Examinations overseas had revealed a palpable liver. Liver function tests performed there were all abnormal but the GI series was negative. The transfer diagnosis was cirrhosis of the liver.

On physical examination he was found to be well-developed, moderately obese, with a palpable liver about two finger-breadths below the right costal margin, firm and non-tender. Otherwise, the physical examination was negative.

Laboratory work showed a prothrombin time of 30 seconds with a control of 20; BSP- 43 percent of dye remaining; icterus index- 14 mg.; thymol turbidity- 18.8 units; cephalin choles-

terol flocculation- 0 at 24 hours, and one plus at 48.

The patient was placed on the combined intensive treatment for cirrhosis including intravenous injections of intraheptol and Solu-B. A liver biopsy was performed and revealed Laennec's cirrhosis. "The lobules vary in size and are surrounded by a large amount of dense, fibrous tissue containing lymphocytes and a few plasma cells. There is an apparent increase in bile duct in the portal connective tissue. The central vein is eccentrically placed or absent in the lobule. The hepatic cells are large with granular type of cytoplasm." This biopsy was performed on 7 September 1948.

On 4 November 1948 a needle biopsy of the liver was performed and microscopic examination was reported as follows: "The small fragment of tissue from the liver contains a moderate number of lobules which vary in size. They are surrounded by a collar of connective tissue containing a few lymphocytes and plasma cells. Within the lobules the architecture is slightly distorted due to fixation and cutting. However, the central veins which are visible appear eccentrically placed. The hepatic cells themselves are of average size, a few containing small clear vacuoles. The cytoplasm of these cells is granular. In comparing this section with the previous section, the inflammatory reaction in the portal spaces appears less marked."

The duration of symptoms before beginning the combined intensive treatment was about one and a half months. The time elapsing after treatment before signs and symptoms improved was four weeks. Laboratory tests at this time in January 1949 showed BSP- 4 percent remaining; icterus index-4; prothrombin time-23 seconds with a control of 21; thymol turbidity-17.8 units; and negative cephalin cholesterol flocculation test.

The amount of time before the liver function tests improved was about five weeks. The time before returning to normal activity was two and a half months. The amount of time before all signs and symptoms disappeared was two months.

Diagnosis: Cirrhosis, Laennec's, moderate, improved.

H.C., a 51 year old white male, was admitted to Brooke General Hospital on 22 June 1948 with a history of mid-abdominal pains of three weeks duration with a similar episode in March 1948. The onset was cramping in nature and was confined primarily to the right lower quadrant. The patient had anorexis but no nausea nor vomiting. His symptoms persisted and he began to notice a weight loss, which he estimated as 20 pounds over the last six months.

Physical examination on admission showed tenderness to palpation over the gall-bladder region. There was evidence of marked weight loss but otherwise the physical examination was negative.

A cholecystographic examination failed to visualize the gall-bladder on repeated examinations. Diagnostic biliary drainage revelaed a normal functioning gall-bladder and chronic gastritis.

On 23 August 1948 exploratory laporotomy was performed. The gallbladder was removed and showed a chronic cholecystitis. The liver was found to be greatly enlarged, chiefly the right lobe. There were many nodules over the entire liver that was visible. There was one large nodule in the right lobe, which was biopsied and histopathological diagnosis was Laennec's cirrhosis. Liver function studies were all within the upper limits of normal.

The patient was placed on the combined intensive treatment for cirrhosis. He showed improvement as manifested by gradual weight gain and stabalized liver function tests until 30 November 1948. By this time his liver had increased to four fingerbreadths below the costal margin, smooth and tender. Needle biopsy of the liver was performed and revealed Laennec's cirrhosis and hepatoma. This diagnosis was confirmed by the Army Institute of Pathology.

On 5 November the patient began to show evidence of collateral venous circulation as manifested by many tortuous veins over the chest and abdomen. By this time his spleen was palpable. X-ray studies of the esophagus revealed more evidence of esophageal varices. By 30 November the liver was palpable as low as the level of the umbilicus and the patient was complaining of right upper quadrant pain and nausea. By 3 December he had pitting ankle edema bilaterally. On 7 December liver function tests showed a definite rise. Thymol turbidity-33.5 units; icterus index-8; prothrombin time-27 seconds with a control of 20; BSP-18 percent dye remaining.

By 12 December 1948, patient was still nauseated and developed diarrhea with five to six stools daily. The stools were black and contained blood. On 12 December he vomited approximately four ounces of fresh blood. The patient continued to slowly go downhill, and in spite of blood transfusions, his red blood count dropped to 2.7 million. On 30 December the patient developed rales in both lung bases and pain in the left chest. He expired on 9 January 1949.

Summary of Autopsy

1. Moderate generalized arteriosclerosis

2. Brown atrophy of the heart

5. Bronchopneumonia-right lower lobe

4. Bilateral hydrothorax

5. Splenomegaly and secondary carcinoma of the spleen

- 6. Secondary carcinoma of the peripancreatic and periaortic lymph nodes
- 7. Moderate varices of the esophagus

8. Secondary carcinoma of the colon

9. Secondary carcinoma of the peritoneum

10. Secondary carcinoma of the liver.

Case 20

J.D., a 29 year old white male, was admitted to the Brooke General Hospital on 18 July 1949 with the chief complaint of malaise, easy fatigueability, jaundice, diarrhea, abdominal discomfort for about one month. The patient was in good health until approximately 22 June 1949 when he noticed the gradual onset of general malaise, easy fatigueability, weakness and about five days later he suddenly became worse. He began having subcostal discomfort aggravated by deep breathing. He developed a fever and was told that he was yellow. He stated that he was 'out of his head' with fever for two or three days. He next developed loose watery bowel movements and had 20 to 25 in one night. He was seen by a local physician and told that he had infectious hepatitis and dysentery. He was given white tablets and put to bed for liver treatment, but continued to have jaundice, diarrhea, and weakness. About three weeks before admission, the patient noticed his stools were clay colored and his urine very dark. For five days prior to admission the patient had a frontal headache. There was no history of previous jaundice. melena, hematemesis, or chills. The patient has been an extremely heavy alcoholic for the last two years, drinking on an average of one quart of whiskey per day.

Physical examination showed a well-developed, well-nourished

white male who weighed 175 pounds, was six foot tall, normal temperature, pulse and blood pressure. Chest was clear, heart not enlarged, abdomen was flat and relaxed. Liver edge was palpable five finger-breadths below the right costal margin. smooth, and slightly tender. The spleen was enlarged three fingerbreadths below the left costal margin. No other masses or organs palpable. There was no icterus. Otherwise, physical examination was negative. The laboratory work showed no parasites in a series of stool examinations. The sedimentation rate was 40 mm., the A/G ratio was 2.0, total protein 7.35. albumen- 4.90gm. and globulin- 2.4 gm. BSP on 25 July showed 12 percent retention, and on 15 August there was 7 percent retention. Cephalin cholesterol flocculation on 22 July was one plus at 24 hours and two plus at 48 hours, and by 18 August it was four plus at 24 hours and four plus at 48. The icterus index on 26 July- 4.0 units, the prothrombin time normal, thymol turbidity- 18.2 units.

The patient was treated with complete bed rest, high caloric, high protein, low fat diet, and multi-vitamins. The patient rapidly became asymptomatic, afebrile and the jaundice, liver enlargement and tenderness gradually disappeared. By 13 August 1949 the patient was allowed up and about the ward which caused no exacerbations or tenderness and by 18 August, the BSP test being 7 percent retention, the patient was discharged to home

Diagnosis: Hepatitis, caused by infection, improved.

This case, we feel, demonstrates the value of the Patek-Post type diet in hepatitis.

Case 21

H.F., a 31 year old white male, was admitted to the Brooke General Hospital on 1 August 1949 with the chief complaint of chills and fever since 22 July 1949, sharp and stabbing pains

in the right upper quadrant.

The patient was feeling perfectly well until 22 July when he had the onset of chills and fever approximately every other day until the present time. He also had nausea with accompanying fever, severe and shaking chills. The patient had had malaria since 1943 with recurrences in 1944, 1946 and 1947, each treated by the patient with quinine. He gave no history of jaundice or liver disease or food intolerance to fats or fried foods. The patient has had constipation since 27 July 1949. He stated that a local physician had found malarial parasites in the blood about one week before admission. He has had no history

of tarry stools or malaise, but does say that cabbage causes indigestion.

Physical examination showed a white male, 70 inches tall, weighing 177 pounds, with a temperature of 101°F, pulse 84 and blood pressure 118/86. No icterus was observed. The abdomen was soft but a definite mass about six by four cm. was palpated along the mid-clavicular line in the right upper quadrant. This mass was tender and it was to this area that the patient finger-pointed his pain. The liver edge was about one finger-breadth below the right costal margin.

Laboratory work on 2 August—WBC-22,750, lymphocytes-28 percent; Hb. 15.5 gms. By 3 August the WBC was 13,600 and on 4 August WBC-9,300. Sedimentation rate was 36 mm in one hour and hematocrit- 46 VPC; serum amylase 32 units; serum bilirubin 0.8 mg. percent; thymol turbidity-0; icterus index-2.0 units; five smears for malaria negative; BSP-8 percent retention; cephalin cholesterol flocculation normal; fecal urobilinogen 95.6 Erlich units per 24 hours; urine urobilinogen 59.4 units in 24 hours; stools, serology, chest x-ray and flat x-ray plate of abdomen were all negative.

When the patient was first admitted he was placed on a liquid diet, given intravenous glucose solution and pantopon for pain. By 3 August his diet was changed to a soft ulcer diet at which time his temperature was receding and the patient became afebrile on the fourth hospital day. Patient was seen in consultation by the surgical service on 4 August who concurred in our impression that the patient had an acute cholecystitis and should have conservative treatment. The mass in the right upper quadrant diminished in size and tenderness and the patient was discharged 11 August 1949 asymptomatic and afebrile.

Diagnosis: Cholecystitis, acute, cause undetermined, improved.

Case 22

W.F., a 21 year old white male, was admitted to the Brooke General Hospital on 27 May 1949 with the chief complaint of recurrent upper abdominal pain and anorexia.

About May 1948 the patient first noted cramping upper abdominal pain usually occurring in the afternoon and lasting about one hour. These attacks would persist for about one week and would be free of symptoms about one week. He had no associated vomiting, nausea, or diarrhea. His bowel movements were small in amount and moved about every three days. When the pain subsided, constipation would also cease. He had had no change in weight in the last year.

Physical examination was negative with the exception of slight soreness on deep pressure over the sigmoid colon and epigastric region. Laboratory work was negative except that stool examinations at the Fourth Army Area Medical Laboratory revealed Endameba Histolytica trophozoites. Proctosigmoidoscopic examination was negative.

The patient was treated with diodoquin 3.2 grains three times a day for ten days, and six subsequent stool examinations

were negative for ova, cysta, or parasites.

On 9 June 1949, procto-sigmoidoscopic examination was normal. The patient at that time was completely asymptomatic and he was discharged from hospital.

Diagnosis: Amebiasis, intestinal, cured.

Case 23

A.G., a 49 year old Latin-American male, was admitted to the Brooke General Hospital on 23 August 1949. At the time of admission, he was in a stuporous condition, unable to answer questions. The history was obtained from his sister as follows: For the past 29 years, the patient had been a heavy consumer of alcohol with poor dietary intake. He first became ill approximately three years ago, consisting of pain in his abdomen and vomiting of blood. He was hospitalized two years ago in San Antonio because of hematemesis. One and a half years ago he was hospitalized at Brooke General Hospital for the same reason. de was told at this last hospitalization that he had a liver disease and should abstain from all alcoholic beverages. This he did not do. He was well until 21 August 1949 when his family noticed a vellowish tint to his skin and sclerae and he became irrational. The patient had mever vomited and had no nausea. No blood had been noticed in the stools nor urine.

Physical examination revealed a 49 year old Latin-American male who was stuperous, incoherent, appeared acutely ill and deeply jaundiced. He was moderately obese with an estimated weight of 220 pounds. Blood pressure— 160/88, pulse—120. The skin was deeply jaundiced. Spider angiomata and large areas of ecchymosis were seen over the upper thorax. Breath sounds were diminished in intensity throughout both lung fields. A systolic murmur was heard at the apex. The abdomen was obese, non-tender and soft. The liver was enlarged four fingers below the right costal border. It was firm and on palpation, felt nodular. There was soft dullness with distant fluid waves of ascites. No laboratory work was done. On 23 August the patient was transferred to the gastroenterology section and 1000 cc. of 10 percent glucose in water intravenous was started. The patient also

received four mg. of vitamin K intramuscularly every three hours. He was placed in an oxygen tent. At 2100 hours the patient became extremely restless and tried to get out of bed and vomited, the vomitus containing about 1000 cc. of coffee ground material; his temperature was 102°F, respirations 28. Eight cc. of paraldehyde was given rectally. Throughout that night the patient vomited small amounts of coffee ground material which contained dark red blood. At 0600 hours on 24 August the patient's pulse suddenly became weak and respirations became deep, noisy and the blood pressure was 132/72. The pulse was 140. The patient expired at 0655 hours.

Summary of Autopsy

Autopsy showed a rupture of the esophageal varices; splenomegaly due to portal obstruction; Laennec's cirrhosis of the liver; solitary cysts of the kidneys; and calculus in the prostate.

Case 24

G.G., a 21 year old white male was admitted to the Brooke General Hospital on 28 December 1948 with a diagnosis of perforated peptic ulcer of the stomach. The patient was admitted first to the surgical service, but they believed he was a case for medical treatment and the patient was transferred to ward 25.

History revealed that he had his onset of acute pain in the epigastric region on 11 November 1948 while overseas. He remained near his bunk about two hours until the pain became so severe it doubled him over. He had no previous symptomatology of peptic ulcer prior to that time. He was taken to a civilian hospital near Liverpool, England and operated on 12 November 1948. His post-operative course was good. His main complication consisted of a pelvic abscess which was against the bladder posteriorly and caused painful urination. The patient kept on a bland diet until admission to Brooke General Hospital.

Physical examination was negative with the exception of a scar on the abdomen and tenderness in the right pelvic gutter.

Laboratory work showed an initial blood count of 12,400 WBC, 14.5 gm. Hb., 46 percent neutrophiles, 40 percent lymphocytes, and 14 percent eosinophiles. Urinalysis showed one to three RBC per HPF, otherwise negative. Serology was negative. A repeat urinalysis was negative. A repeat blood count revealed 5.0 million RBC, 10,500 WBC, 50 percent neutrophiles, 43 percent

lymphocytes, and 7 percent eosinophiles. GI series on 10 January 1949 showed esophagus and stomach to be normal but there was a deformity of the duodenal cap and a small ulcer crater in the site of the operative repair. By 1 February 1949 the crater was no longer demonstrable. Fractional gastric analysis revealed a Grade II gastric hyperacidity. The patient was placed on a convalescent ulcer diet and resinat therapy. He became asymptomatic very shortly.

Diagnosis: Perforation of the stomach due to peptic ulcer, moderate, chronic, improved.

Ulcer, peptic, of duodenum, moderate, chronic, improved.

Case 25

J.G., a 72 year old white male, was admitted to the Brooke General Hospital on 9 February 1949 with the chief complaint of pain in both shoulders with radiation to both elbows, pain in the legs, and shortness of breath.

About two years before admission the patient began having pain in both shoulders. This apparently was not related to exercize, but walking two blocks caused pain in the legs and kidney region and the pain left after resting about four minutes. The leg pain was from the knees down. Occasionally, he had leg cramps at night causing him to get out of bed. If his legs were kept warm, the pain did not occur. The patient had had shortness of breath for the last three years with increasing severity. Three weeks before admission this became severe and he had to sit up in bed to breathe. He has had no precordial chest pain. The patient has had a chronic cough for the last three years but no sputum. About three years ago he coughed up a teaspoonful of blood and his physician said it was from his lungs. The patient gave a history of heavy alcoholic consumption.

Physical examination revealed a man of stated age. Fundiscopic examination revealed a Grade I to Grade II arteriosclerosis with silver wire reflexes from the arteries. The chest revealed emphysema with fixation of the thoracic cony cage with anterior posterior diameter increased and it was necessary for him to use accessory muscles for respiration. He also had moist rales in both bases not clearing on coughing or deep inspiration. An aortic soft diastolic murmur was heard best at the left interspace at the left side of the sternum, also apical murmurs at the PMI, not transmitted. The

abdomen was obese, pendulous, and the liver was down about three finger-breadths, slightly tender, not nodular and the gall-bladder was not palpable. There were no other masses felt, except the spleen which could be palpated two inches below the left costal border. The skin showed a sclerotic veins bilaterally with dependent skin changes on the left ankle.

The laboratory work on admission showed 3,800 WBC.with normal differential count; 2.67 million RBC; 7.5 gm. Hb... icterus index- 25 units; prothrombin time normal; thymol turbidity- 23 units; acid phosphatase- 1.02 units; alkaline phosphatase- 6.0 units; blood urea nitrogen-32 mg., and a repeat was 51 mg percent. EKG was normal. Sternal bone marrow showed Hodgkin's disease. The bone marrow was aspirated from the second interspace through a Turkel trephine. Microscopic examination: " Smears stained with Wright's stain showed a bone marrow which is reduced in cellular elements. Granulopoiesis appeared normal. Erythropoiesis is shifted to the left. Macrophages are increased and actively phagocytic. There is a noticeable increase in plasma cells, some showing vacuolated cytoplasm. There are frequent atypical cells which may be described as hypertrophical reticulum cells not unlike Reed-Sternberg's cells. Impression gained from blood and bone marrow was Hodgkin's disease."

The patient was initially admitted to the cardiac ward because of pain radiating to both elbows and ankles, and edema. He did not respond to cardiac therapy. Because of the liver findings, he was transferred to ward 25. The patient became jaundiced five days after admission to the hospital and it was the impression that the dependent edema was secondary to cirrhosis of the liver and/or hepatitis.

After transfer to ward 25 he became progressively jaundiced and went into coma. The patient never recovered from this coma and expired 20 February 1949.

Clinical Diagnosis:

- 1. Hodgkin's disease.
- 2. Laennec's cirrhosis
- 3. Chronic cholecystitis.
- 4. Arteriosclerotic heart disease.

Summary of Autopsy:

- 1. Laennec's cirrhosis of the liver.
- 2. Arteriosclerotic heart disease.
- 3. Chronic cholecystitis with cholelithiasis in the common duct.
- 4. Arteriolar nephrosclerosis, bilateral.

G.H., a 54 year old white male, was first admitted to the closed neuropsychiatric section, Brooke General Hospital on 9 August 1949 with a diagnosis of psychosis of two or three months duration. The patient's only complaint was weakness. His history was considered very inadequate and unreliable due to the mental status of the patient. He had the usual child-hood diseases. No serious adult illnesses were ascertained. The patient had had two nervous breakdowns within the past three years, the last one three months prior to admission.

Physical examination revealed a well-developed, poorly nourished white male of stated age who was rather flippant and occasionally hostile. Pulse- 132. Blood pressure normal. Pupils reacted poorly to light, skin was very pale and in

some areas had a yellowish tint.

Laboratory work showed 1.19 million RBC with 5.0 gm. Hb. Hematocrit-15 VPC. Cardiolipin microflocculation and cardiolipin complement fixation were positive. Spinal fluid revealed a cell count of 1 lymphocyte, total protein-74.5 mg. percent, positive globulin. colloidal gold curve-0011100000. Spinal fluid serology positive in one half to one cc. drops. Sputum negative for acid fast bacilli. Chest x-ray showed a mass in the left lung field adjacent to the bifurcation of the left main stem, measuring 3.5 by 6.0 cm. in diameter which was obstructing the lower bronchus but had apparently deviated the lower bronchus medially. GI series 18 August 1949 showed a large ulcer crater in the mid third of the lesser curvature of the stomach. It did not appear malignant. Sternal bone marrow aspiration 15 August revealed hypoplasia and a questionable tumor cell in the bone marrow. Biopsy of the lymph node in the left temporal area 18 August revealed secondary undifferentiated carcinoma. Gastroscopic examination 19 August gave the opinion that the large crater seen was benign.

Because of marked anemia, he was given 2500 cc. of blood, and because of a positive serology he received 10 million units of penicillin. On 6 September the patient was presented to the Tumor Board and a diagnosis of primary carcinoma of the lung, generalized metastasis and cerebro-vascular lues was made. Recommendation was palliative therapy and symptomatic treatment was made. At 0345 hours 3 September 1949 the patient developed marked respiratory distress, his color was ashen grey, pulse 104, respiration 84, blood pressure- 100/50. Gurgling noises were heard in the throat and moist rales in the lung fields. The patient was placed in an oxygen tent. Despite all efforts of therapy, the patient expired 0445 hours 9 september 1949.

Clinical Disgnosis:

- 1. Adenocarcinoma of the bronchus, with generalized metastasis.
- 2. Gastric ulcer.
- 3. Meningovascular syphilis.

Summary of Autopsy:

- 1. Bronchogenic carcinoma, left lung.
- 2. Secondary carcinoma, right lung.
- 3. Metastatic carcinoma to the tracheobronchial nodes.
- 4. Peptic ulcer of stomach.
- 5. Secondary carcinoma, metastatic, of the left kidney and diaphragm.

Case 27

W.J., a 60 year old white male, was admitted to the Brooke General Hospital on 19 May 1949 with chief complaints of headache, backache, and burning in the stomach.

In April 1945 he first noticed a swelling in the upper abdomen associated with epigastric cramping pain usually followed by burning in the same area. The pain usually occurred about nine to ten o'clock at night but never awakened him. He complained of soreness in the epigastric region and headache occurring twice weekly. In April 1945 he was admitted to Brooke General Hospital on the surgical service, remaining about three weeks and told he had a "hole in his stomach". On a milk diet for seven days he became asymptomatic and had no operation. He had no further trouble until May 1948 when swelling in the epigastrium returned with pain and burning causing him to quit his work. In May 1949 he began having chills and fever with profuse perspiration, and severe shaking. The fever rose to 104°F on one occasion. The chills lasted about two weeks and disappeared without medication. Because of recurrence of abdominal swelling, upper abdominal pain, and burning he was admitted to ward 25.

Physical examination showed a well healed hernioplasty scar in the left lower quadrant. Moderate tenderness to deep pressure just below the umbilious was elicited. Otherwise negative.

Laboratory work showed slight kyphosis of the lower dorsal and lordosis of the lumbar spine. GI series showed a short esophagus. There was a fairly large herniation of the stomach through the diaphragm. The duodenal cap was normal. No retention at five hours. These findings were consistent with diaphragmatic hernia.

The patient was seen in surgical consultation and it was

believed he should have repair of the disphragmatic hernia but the patient refused surgery at that time.

He was treated with a convalescent ulcer diet, bellafoline, and during treatment remained asymptomatic except for occasional bloating and gas pains. Patient was sent home with frequent small feedings and advised to sleep with his head elevated.

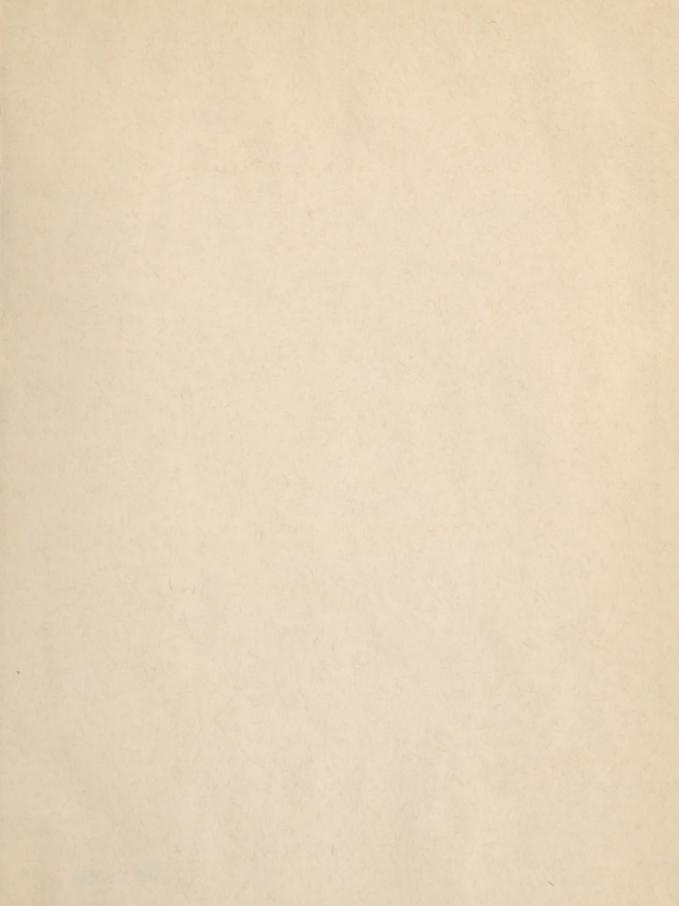
Diagnosis: Hernia, diaphragmatic.

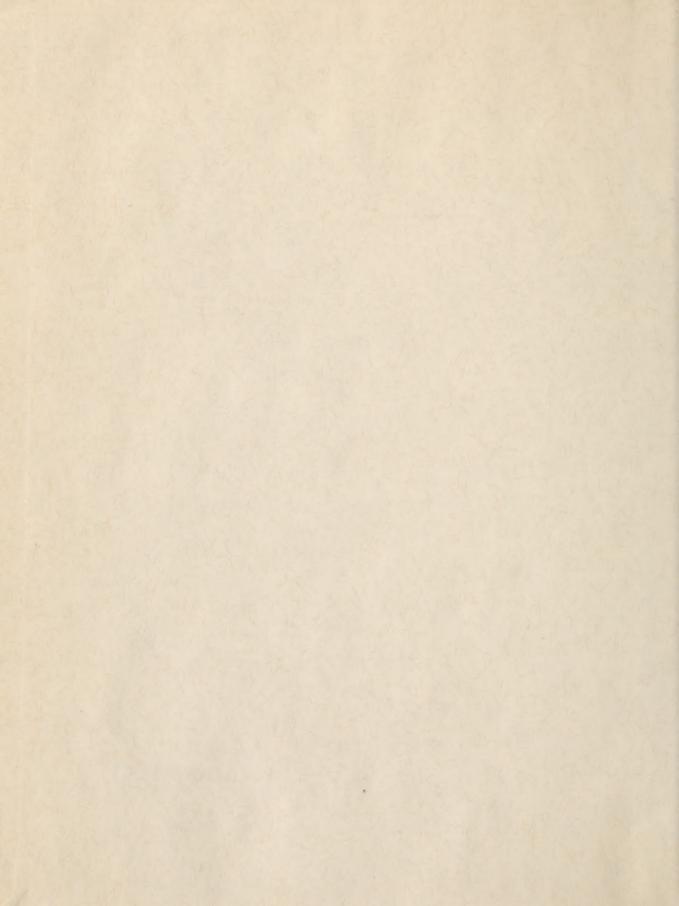
Kyphosis of the spine, cause undetermined. Scoliosis of the spine, cause undetermined.



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